

Appl. No. : 09/111,123

Filed : 7/6/1998

REMARKS

Claims 1 - 7 stand rejected under 35 USC 102(a) over Liu et al ("Liu"). However, applicant respectfully disagrees. Liu is an Abstract which describes, without any specificity, a strategy of genetically grafting into the heavy chain of humanized anti-human Fc γ RI mAB22 peptides to produce two constructs, Fab22-TT830 and Fab22-TT33S, to increase presentation of TH epitopes to increase the immunogenic potency of peptide-based vaccines. The constructs then bind to Fc γ RI by their intact Fab region, thereby containing the peptide in place of the Fc region. Liu then mentions at its end that Fc γ RI targeting as taught in Liu was able to significantly increase the effectiveness of antigenic peptide presentation and thus: "[t]his suggests that targeted presentation of antagonistic peptides could lead promising Ag-specific therapies for T cell-mediated autoimmune disorders."

Liu simply speculates that such an approach as taught by Liu *might* be useful in treatment of T cell mediated autoimmune disorders. However, the Liu abstract provides absolutely no evidence, data or conclusions demonstrating that it in fact *would* be useful to prevent activation or downregulate T cells responsible for an autoimmune disorder. Thus the reference is not anticipatory under 35 USC 102(a) of the claimed invention. To be an anticipatory, the "identical invention must be shown in as complete detail as is contained in the ... claim." Richardson v. Suzuki Motor Co., 868 F.2d 1226, 1236, 9 USPQ2d 1913, 1920 (CAFC 1989). There is no teaching in Liu of a fusion protein for alleviating symptoms of an autoimmune disorder by deactivation of T cells.

The Liu construct is made so that the intact Fab region binds to the Fc γ RI and contains the peptide in place of the Fc region which is entirely different in how the applicant's construct is made and functions. Liu's construct is potentially dangerous to treat an autoimmune disorder because it is based on antigen-antibody affinity and thus the antibody of Liu has a high affinity to the Fc receptor and could block the receptor from physiologic use for immune defense and phagocytosis of immune complexes and bacteria. Even worse, Liu's construct could activate APCs and induce costimulatory molecules which would facilitate inflammation and exacerbate the autoimmune disorder rather than suppress the autoimmune disorder. Because the Liu

Appl. No. : 09/111,123

Filed : 7/6/1998

construct involves antigen-antibody affinity and there is such a high affinity of the Liu construct for Fc receptors, it is possible that the Liu construct would activate cells that are not APCs and trigger non-specific polyclonal activation and possibly multiple disorders. The Liu Abstract is not a proper prior art reference in that Liu fails to teach all of the aspects of the claimed invention.

Liu is not an enabling reference for the purpose that the Examiner is asserting. Per MPEP 2121.01, "in determining that quantum of prior art disclosure which is necessary to declare an applicant's invention 'not novel' or 'anticipated' within section 102, the standard test is whether a reference contains an enabling disclosure..." In re Hoeksema, 399 F.2d. 269, 158 USPQ 596 (CCPA 1968). Because Liu is not a patent, there is no presumption that its fusion proteins are operable for the purpose of deactivating T cells. And as previously stated, there is no evidence that the Liu fusion proteins would be useful to deactivate T cells, only the mere suggestion in the Abstract. A reference contains an enabling disclosure only if the public was in possession of the claimed invention before the date of the invention. MPEP 2121.01. In this case, there is no evidence that Liu put in possession of the public fusion proteins for treating autoimmune disorders by deactivating autoreactive T cells.

Neither would the claimed invention be obvious over Liu for many of the reasons already stated. This is an entirely different construct than that of the claimed invention and operates in a completely different manner. Liu actually teaches away from the claimed invention. The teachings of the Liu Abstract are neither anticipatory nor obviating and withdrawal of the rejection is respectfully requested.

Claims 1 - 7 stand rejected under 35 U.S.C. 112(1) for reasons stated of record. The claims have been amended to specify that the inhibition of T cell activation are those T cells responsible for the underlying autoimmune disorder. Thus, the rejection is believed to be overcome and withdrawal of the rejection is respectfully requested.

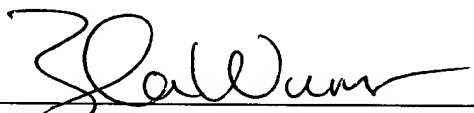
Claims 1 - 7 stand provisionally rejected under the judicially created doctrine of obviousness-type double patenting over claims 4, 6, 9, 11, 24, 26, 27, 29, 66 - 70 and 72 - 73 of U.S. Patent Application Serial No. 08/779,767. Because the rejection is provisional and the claims of each patent have not yet been allowed, it is impossible at this time to evaluate whether the allowed/issued claims of the present invention are in fact obvious over the allowed/issued

Appl. No. : 09/111,123
Filed : 7/6/1998

claims of copending application Serial No. 08/779,767. Applicant's attorney respectfully requests the right to revisit the issue of filing a terminal disclaimer at the time the present claims are formally allowed.

Applicant hereby requests a three month extension of time. A version to show changes to claim 1 is attached as Exhibit A. If there are any questions concerning this response, applicant's attorney can be reached at the telephone number stated below.

Dated: 7/11/03

By: 
John Wurst
Registration No. 40,283
(858) 410-5174

Appl. No. : 09/111,123

Filed : 7/6/1998

VERSION WITH MARKINGS TO SHOW CLAIM CHANGES

1. A fusion protein for the alleviation of symptoms associated with an autoimmune disorder comprising an immunoglobulin or portion thereof linked to one or more T cell receptor antagonists wherein said immunoglobulin or portion thereof is capable of binding to an Fc receptor and being endocytosed by an antigen presenting cell to present said one or more T cell receptor antagonists in association with endogenous MHC Class II molecules, thereby preventing [T cell] activation of T cells specific for said T cell receptor antagonist.